Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
S1	169	"apolipoprotein A-1"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:38
S2	78	"apolipoprotein A-1" and phospholipid	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/01/10 14:12
S3	15	"apolipoprotein A-1" same phospholipid	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/01/10 14:12
S4	5	"637313".ap.	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/01/13 11:44
S5	2	"5861267".pn.	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/01/13 16:23
S6	2	"4762914".pn.	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/01/13 16:45
S7	2	"5861267".pn.	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/01/13 17:06
S8	547	"apolipoprotein A-1" or apoA-I	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:39
S9	429	("apolipoprotein A-1" or apoA-I)and cholesterol	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:39
S10	381	("apolipoprotein A-1" or apoA-I)and cholesterol and HDL	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:39
S11	274	("apolipoprotein A-1" or apoA-I) same cholesterol and HDL	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:40
S12	223	("apolipoprotein A-1" or apoA-I) same cholesterol and HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:40

S13	99	("apolipoprotein A-1" or apoA-I) adj10 cholesterol and HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:40
S14	74	("apolipoprotein A-1" or apoA-I) adj5 cholesterol and HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:41
S15	5	("apolipoprotein A-1" or apoA-I) adj cholesterol and HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:41
S16	54	("apolipoprotein A-1" or apoA-I) adj3 cholesterol and HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:41
S17	22	("apolipoprotein A-1" or apoA-I) adj2 cholesterol and HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:42
S18	188	("apolipoprotein A-1" or apoA-I) same cholesterol same HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:42
S19	91	("apolipoprotein A-1" or apoA-I) same cholesterol same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:42
S20	91	("apolipoprotein A-1" or apoA-I) same cholesterol same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:08
S21	3	("apolipoprotein A-1" or apoA-I) same cholesterol with decrease same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:45
S22	0	("apolipoprotein A-1" or apoA-I) same lowering with cholesterol same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:45
S23	3	("apolipoprotein A-1" or apoA-I) same decrease with cholesterol same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:45
S24	10001	(lowering or decreasing or removing) with cholesterol	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:46

S25	91	(lowering or decreasing or removing) with cholesterol same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:47
S26	830	(lowering or decreasing or removing) with cholesterol same ApoA-I HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:48
S27	1	(lowering or decreasing or removing) with cholesterol same ApoA-I same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 13:48
S28	41	("apolipoprotein A-1" or apoA-I) same cholesterol with free same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:09
S29	0	("apolipoprotein A-1" or apoA-I) same "cholesterol free" same HDL same phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:09
S30	0	("apolipoprotein A-1" or apoA-I) same "cholesterol free" same HDL and phospholipids	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:10
S31	0	("apolipoprotein A-1" or apoA-I) same "cholesterol free"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:10
S32	33	("apolipoprotein A-1" or apoA-I) and "cholesterol free"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:10
S33	0	("apolipoprotein A-1" or apoA-I) same "cholesterol free"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/08/25 14:10

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INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
       AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,
       BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
       CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 15:54:19 ON 28 AUG 2005

74 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
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=> ("apolipoprotein A-1" or apoA-I) and "cholesterol free"
         14   FILE BIOSIS
  13 FILES SEARCHED...
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          8   FILE CABA
          9   FILE CAPLUS
  19 FILES SEARCHED...
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          4   FILE ESBIOBASE
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         11   FILE MEDLINE
          1   FILE PASCAL
          2   FILE PROMT
  60 FILES SEARCHED...
          4   FILE SCISEARCH
          4   FILE TOXCENTER
         25   FILE USPATFULL
          2   FILE USPAT2
  68 FILES SEARCHED...
  72 FILES SEARCHED...

  16 FILES HAVE ONE OR MORE ANSWERS,   74 FILES SEARCHED IN STNINDEX

L1   QUE ("APOLIPOPROTEIN A-1" OR APOA-I) AND "CHOLESTEROL FREE"

=> d rank
F1          25   USPATFULL
F2          14   BIOSIS
F3          11   MEDLINE
F4           9   CAPLUS
F5           9   EMBASE
F6           8   CABA
F7           4   ESBIOBASE
F8           4   SCISEARCH
F9           4   TOXCENTER
F10          2   BIOTECHNO
F11          2   PROMT
F12          2   USPAT2
F13          1   FSTA
F14          1   IFIPAT
F15          1   LIFESCI
F16          1   PASCAL


=> file biosis medline caplus embase caba
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=> ("apolipoprotein A-1" or apoA-I) and "cholesterol free"
L2 51 ("APOLIPOPROTEIN A-1" OR APOA-I) AND "CHOLESTEROL FREE"

=> dup remove
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PROCESSING COMPLETED FOR L2
L3 19 DUP REMOVE L2 (32 DUPLICATES REMOVED)

=> d ti 1-19

L3 ANSWER 1 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Influence of **ApoA**-I structure on the ABCA1-mediated efflux of cellular lipids.

L3 ANSWER 2 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
TI Response of ApoA-IV in pigs to long-term increased dietary oil intake and to the degree of unsaturation of the fatty acids.

L3 ANSWER 3 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 2
TI Hypocholesterolaemic effects of an ethanol precipitate of Kabosu juice in stroke-prone spontaneously hypertensive rats fed a **cholesterol-free** diet.

L3 ANSWER 4 OF 19 CAPLUS COPYRIGHT 2005 ACS on STN
TI Interaction of apolipoprotein A-I with lecithin-cholesterol vesicles in the presence of phospholipase C

L3 ANSWER 5 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Dietary taurine enhances cholesterol degradation and reduces serum and liver cholesterol concentrations in rats fed a high-cholesterol diet.

L3 ANSWER 6 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 3
TI Third trimester fetal growth and measures of carbohydrate and lipid metabolism in umbilical venous blood at term.

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on STN DUPLICATE 4
TI Third trimester fetal growth and measures of carbohydrate and lipid metabolism in umbilical venous blood at term.

L3 ANSWER 8 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 5
TI Effect of LpA-I composition and structure on cholesterol transfer between lipoproteins.

L3 ANSWER 9 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 6
TI Apolipoprotein mRNA in liver and intestine of rats is affected by dietary beet fiber or cholestyramine.

L3 ANSWER 10 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Role of elevated lecithin:cholesterol acyltransferase and cholesteryl ester transfer protein activities in abnormal lipoproteins from proteinuric patients.

L3 ANSWER 11 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 7
TI EFFECTS OF LIPID TRANSFER PROTEIN ON PLASMA LIPIDS APOLIPOPROTEINS AND METABOLISM OF HIGH-DENSITY LIPOPROTEIN CHOLESTERYL ESTER IN THE RAT.

L3 ANSWER 12 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 8
TI CHARACTERIZATION OF PLASMA LIPOPROTEINS OF GRAIN-FED AND CHOLESTEROL-FED WHITE CARNEAU AND SHOW RACER PIGEONS.

L3 ANSWER 13 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 9
TI THE CONTRASTING EFFECT OF DIETARY PHOSPHATIDYL ETHANOLAMINE AND PHOSPHATIDYL CHOLINE ON SERUM LIPO PROTEINS AND LIVER LIPIDS IN RATS.

L3 ANSWER 14 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 10
TI LIPO PROTEIN LIPID AND PROTEIN RESPONSES TO DIETARY FAT AND DIABETES IN RATS.

L3 ANSWER 15 OF 19 CABA COPYRIGHT 2005 CABI on STN
TI Effects of dietary proteins on the intestinal synthesis and transport of cholesterol and apolipoprotein A-I in rats.

L3 ANSWER 16 OF 19 MEDLINE on STN
TI Effects of dietary trans-fat on biliary and fecal steroid excretion and serum lipoproteins in rats.

L3 ANSWER 17 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 11
TI EFFECTS OF BETA SITO STEROL ON THE CONCENTRATIONS OF SERUM AND LIVER CHOLESTEROL AND SERUM APO LIPO PROTEINS IN RATS FED BUTTER FAT.

L3 ANSWER 18 OF 19 MEDLINE on STN DUPLICATE 12
TI Serum and liver cholesterol levels of rats and mice fed soy-bean protein or casein.

L3 ANSWER 19 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 13
TI EFFECTS OF NICOTINIC-ACID THERAPY ON PLASMA HIGH DENSITY LIPO PROTEIN SUBFRACTION DISTRIBUTION AND COMPOSITION AND ON APO LIPO PROTEIN A METABOLISM.

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=> d ab bib 2,6, 8, 13, 10

L3 ANSWER 2 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
AB ApoA-IV is a protein constituent of HDL particles; the gene coding for it is a member of the **ApoA-I**-ApoC-III-ApoA-IV cluster.
To investigate the effects of the quantity and the degree of saturation of dietary lipid on the long-term response of this Apo, and on the hypothetical coordinated regulation of the cluster in vivo, pigs were fed isoenergetic, **cholesterol-free**, low-lipid or
lipid-enriched diets (containing either extra olive oil (rich in MUFA) or sunflower oil (rich in n-6 PUFA)) for 42 d. In animals fed on the control diet, ApoA-IV was mainly associated with plasma lipoproteins. An increase in plasma ApoA-IV concentration, mainly in the lipoprotein-free fraction, was induced by the lipid-enriched diets, independent of the degree of saturation of the fatty acids involved. The latter diets also led to increases in hepatic **ApoA-I**, ApoA-IV and ApoC-III mRNA
levels, more so with the sunflower oil-rich diet. The present results show that porcine plasma ApoA-IV levels and their association with lipoproteins are very sensitive to increases in dietary lipids, independent of the degree of fatty acid saturation. Furthermore, hepatic expression of RNA appears to be coordinated along with that of the other members of the gene cluster.
AN 2005:49025 BIOSIS
DN PREV200500050521
TI Response of ApoA-IV in pigs to long-term increased dietary oil intake and to the degree of unsaturation of the fatty acids.
AU Navarro, Maria A.; Acin, Sergio; Carnicer, Ricardo; Guzman-Garcia, Mario A.; Arbone-Mainar, Jose M.; Surra, Joaquin C.; Cebrian, Jose A.; Arnal, Carmen; Isabel, Beatriz; Lopez-Bote, Clemente J.; Osada, Jesus [Reprint Author]
CS Dept Bioquim and Biol Mol and Celular, Univ Zaragoza, E-50009, Zaragoza, Spain
Josada@unizar.es
SO British Journal of Nutrition, (November 2004) Vol. 92, No. 5, pp. 763-769. print.
CODEN: BJNUAV. ISSN: 0007-1145.
DT Article
LA English
ED Entered STN: 26 Jan 2005
Last Updated on STN: 26 Jan 2005

L3 ANSWER 6 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 3
AB Aim-To compare measures of carbohydrate and lipid metabolism in umbilical venous blood after birth at term in pregnancies with normal and retarded fetal growth during the third trimester. Methods-Three groups of pregnancies reaching term, in which fetal growth had been prospectively monitored by repeated ultrasound measurements during the third trimester, were studied. Sequential fetal abdominal circumference measurements remained above the 10th centile in 42 (normal size, normal growth group),

below the 10th centile but did not depart further than 1.5 SD (small, normal growth group), or below the 10th centile and subsequently fell away by more than 1.5 SD before delivery (small, growth retarded group). Birthweight, neonatal morphometric measures (ponderal index, mid arm:head circumference ratio, subscapular and triceps skinfold thickness), umbilical venous blood concentrations of glucose, insulin, pro-insulin, des 31,32 proinsulin, total **cholesterol**, **free** cholesterol, cholesterol ester, triglycerides, lipoprotein (a), **apolipoprotein A-1** and apolipoprotein B were measured. Results-The median birthweight of the three groups was significantly different (3570, 2569, and 2277 g, respectively). Median values of ponderal index and mid arm:head circumference ratio were significantly lower in the small, growth retarded group and did not differ between the small and normal size groups with normal growth. Both groups with small fetuses had significantly lower mean glucose and cholesterol ester concentrations, and higher mean free cholesterol:cholesterol ester ratios, compared with the normal size, normal growth group. The group showing fetal growth retardation had mean total cholesterol and mean cholesterol ester concentrations that were significantly lower than those of both the other two groups. Mean des 31,32 proinsulin concentrations were low in both groups of small fetuses, but only significantly so in the group without fetal growth retardation. Mean insulin, proinsulin, free cholesterol, triglycerides, lipoprotein(a), **apolipoprotein A-1**, apolipoprotein B concentrations and the ratio of A-1:B were similar in all three groups. Conclusion-The similarity in the umbilical venous blood carbohydrate and lipid profile at term between pregnancies with documented third trimester fetal growth retardation and those with "genetically" small babies argues against a major role for intrauterine nutritional deprivation as a cause for the association between birthweight and subsequent adult disease.

AN 1997:129144 BIOSIS
DN PREV199799420957
TI Third trimester fetal growth and measures of carbohydrate and lipid metabolism in umbilical venous blood at term.
AU Spencer, John A. D. [Reprint author]; Chang, Tou C.; Crook, David; Proudler, Anthony; Felton, Carl V.; Robson, Stephen C.; Hauesler, Martin
CS Dep. Obstet. Gynaecol., Northwick Park Hosp., Watford Rd., Harrow, Middlesex HA1 3UJ, UK
SO Archives of Disease in Childhood, (1997) Vol. 76, No. 1 FETAL AND NEONAT. ED., pp. F21-F25.
CODEN: ADCHAK. ISSN: 0003-9888.
DT Article
LA English
ED Entered STN: 25 Mar 1997
Last Updated on STN: 25 Mar 1997

L3 ANSWER 8 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 5
AB The effect of high density lipoprotein composition on the rates of unesterified cholesterol exchange between low density lipoproteins (LDL) and well-defined homogeneous discoidal lipoproteins (LpA-I) reconstituted with phosphatidylcholine, cholesterol, and apolipoprotein A-I (**apoA-I**) has been investigated. LpA-I containing cholesterol and 2,3, and 4 **apoA-I** molecules per particle differed in their ability to accept or donate cholesterol. A significant cholesterol exchange occurs between LDL and Lp2A-I (7.8 and 9.6 nm), while there is little or no cholesterol exchange detectable between LDL and Lp3A-I (10.8 and 13.4 nm) and Lp4A-I (17.0 nm) complexes. The cholesterol transfer from LDL to the **cholesterol-free** Lp2A-I (9.6 nm), Lp3A-I (13.4 nm), and Lp4A-I (17.0 nm) particles also shows significant cholesterol transfer to Lp2A-I, while there is no detectable transfer to Lp3- and 4A-I particles. The rates of cholesterol transfer to **cholesterol-free** and cholesterol-containing Lp2A-I appear to differ significantly. Cholesterol

transfer from LDL to **cholesterol-free** Lp2A-I is zero order with respect to acceptor concentrations when the Lp2A-I/LDL ratio is above 10. Transfer rates from LDL to **cholesterol-free** Lp2A-I are faster for the smaller Lp2A-I (8.5 nm) than to the larger Lp2A-I (9.7 nm) and exhibit half-times (t-1/2) at 25 degree C of 4.0 and 5.3 h, respectively. In contrast, cholesterol transfer from LDL to cholesterol-containing Lp2A-I remains dependent upon acceptor concentrations to an acceptor/donor particle ratio of 80. In addition, transfer from LDL to cholesterol-containing Lp2A-I is faster to the 9.6 nm than to 7.8 nm particles, with t-1/2 of 1.4 and 2.3 h, respectively. The rates of cholesterol transfer from Lp2A-1 to LDL are higher than in the opposite direction, in particular for the small Lp2A-I (7.8 nm), which has a t-1/2 of approximately 50 min. The results show that changes in the composition and structure of **apoA-I**-containing particles have a significant effect on inter-lipoprotein exchange of cholesterol. This suggests that the kinetics of cholesterol transfer to and from reconstituted discoidal LpA-I particles cannot be fully explained by passive aqueous diffusion.

AN 1995:166388 BIOSIS
DN PREV199598180688
TI Effect of LpA-I composition and structure on cholesterol transfer between lipoproteins.
AU Meng, Qiang-Hua [Reprint author]; Sparks, Daniel L.; Marcel, Yves L.
CS Lipoproteins Atherosclerosis Group, Univ. Ottawa Heart Inst., Ottawa, ON K1Y 4E9, Canada
SO Journal of Biological Chemistry, (1995) Vol. 270, No. 9, pp. 4280-4287. CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 26 Apr 1995
Last Updated on STN: 27 Apr 1995

L3 ANSWER 13 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 9

AB The effect of dietary phosphatidylethanolamine (PE) and phosphatidylcholine (PC) added to **cholesterol-free** semipurified diet on serum lipoprotein and hepatic and fecal lipids was compared to the effect on rats fed soybean oil (controls). The dietary PE, but not PC, caused a decrease in serum cholesterol, phospholipid, apolipoprotein A-I (**apoA-I**) and apoE and an increase in high MW apoB. The simultaneous addition of PC and ethanolamine also decreased serum **apoA-I** and cholesterol. The distribution patterns of phospholipid subclasses in the liver and fatty acid composition of hepatic and plasma phospholipids were also altered by dietary PE. Both PE and PC increased to a similar extent the excretion of fecal neutral steroids and hepatic 3-hydroxy-3-methylglutaryl-CoA reductase activity compared to the controls. The PE in the soybean phospholipid preparation is responsible for the alterations of profiles of serum lipids and apoproteins in rats.

AN 1984:236571 BIOSIS
DN PREV198477069555; BA77:69555
TI THE CONTRASTING EFFECT OF DIETARY PHOSPHATIDYL ETHANOLAMINE AND PHOSPHATIDYL CHOLINE ON SERUM LIPO PROTEINS AND LIVER LIPIDS IN RATS.
AU IMAIZUMI K [Reprint author]; MAWATARI K; MURATA M; IKEDA I; SUGANO M
CS LAB NUTRITION CHEM, DEP FOOD SCIENCE AND TECH, SCH AGRIC, KYUSHU UNIV, FUKUOKA 812, JAPAN
SO Journal of Nutrition, (1983) Vol. 113, No. 12, pp. 2403-2411. CODEN: JONUAI. ISSN: 0022-3166.
DT Article
FS BA
LA ENGLISH

L3 ANSWER 10 OF 19 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

AB Lecithin:cholesterol acyltransferase (LCAT) and cholesteryl ester transfer protein (CETP) are key factors in the esterification of free cholesterol, and the distribution of cholesteryl ester among lipoproteins in plasma. Alterations in these processes may play a role in the lipoprotein abnormalities associated with glomerular proteinuria. The activities of LCAT and CETP were measured using excess exogenous substrate assays in nine patients with nephrotic-range proteinuria and in 18 matched controls. The proteinuria-lowering effect of four weeks of angiotensin converting enzyme (ACE) inhibition with enalapril was also studied. Plasma very low lipoprotein and low density lipoprotein (VLDL and LDL) cholesterol, triacylglycerol and apolipoprotein B levels were significantly elevated in the patients compared with controls. High density lipoprotein (HDL) total **cholesterol**, **free** cholesterol, cholesteryl ester and the free cholesterol/cholesteryl ester ratio in HDL were lower. Total plasma **apolipoprotein A-1** was normal. Plasma LCAT and CETP activities were elevated in the patients by 30% (P lt 0.01) and by 39% (P lt 0.01), respectively, and were both inversely related to serum albumin. VLDL and LDL cholesterol levels were positively related to LCAT and CETP activities, whereas the HDL free cholesterol content was inversely related to LCAT activity. ACE inhibition resulted in a 40% reduction of proteinuria, a partial normalization of LCAT activity, and a decrease in VLDL and LDL cholesterol. In conclusion, elevated activities of LCAT and CETP may provide a mechanism that contributes to the low proportion of cholesterol in HDL relative to that in VLDL and LDL, as well as to the compositional changes of HDL seen in glomerular proteinuria. Such abnormalities could contribute to accelerated development of atherosclerosis in proteinuric states.

AN 1993:395924 BIOSIS
DN PREV199396071224

TI Role of elevated lecithin:cholesterol acyltransferase and cholesteryl ester transfer protein activities in abnormal lipoproteins from proteinuric patients.

AU Dullaart, Robin P. F. [Reprint author]; Gansevoort, Ron T.; Dikkeschei, Bert D.; De Zeeuw, Dick; De Jong, Paul E.; Van Tol, Arie
CS Dep. Endocrinol., Univ. Hosp. Groningen, P.O. Box 30.001, 9700 RB Groningen, Netherlands

SO Kidney International, (1993) Vol. 44, No. 1, pp. 91-97.
CODEN: KDYIA5. ISSN: 0085-2538.
DT Article
LA English
ED Entered STN: 23 Aug 1993
Last Updated on STN: 24 Aug 1993